STAT

ELECTROENCEPHALOGRAPHIC EXAMINATION IN ELECTROSHOCK TREATMENT OF SCHIZOPHRENIA

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Although psychiatric literature is already rather well-supplied with communications on the treatment of schizophrenia by electroshock, investigations of the electrical activity of the brain during such treatments have been conducted only recently. Between 1942 and 1947 several accounts were published concerning electroencephalographic investigations conducted during electro-shock therapy of schizophrenia. Many authors note during such treatments the appearance of disrhythms (of delta waves) in the wake of a series of electroshock convulsions. This is observed more frequently in persons who have slow alpha rhythms preceding the shock than in persons who have fast ones. Such changes in the electroencephalograms of patients have been used as a basis for criticizing methods of applying electroshock. Thus, Proktor and Gudvin in their last work published in 1945 compared the effects of convulsions produced by alternating current with the action of interrupted direct current on the basis of electroencephalograms. They came to the conclusion that an increase in slow activity is

is more marked with the use of alternating current than with that of intermittent direct current. This served as the basis for the authors' preference of the latter method because it offered more supporting evidence for a prognosis. For example, no perceptible disrhythm is observed in cases of complete remission. A partial remission is accompanied by small activity, which in turn is less marked than with cases showing no therapeutic effect. However, these conclusions are debatable inasmuch as the authors do not indicate whether the number of shocks was the same for both groups. A comparison of the effect of both types of electrical current is also not convincing inasmuch as both types of current were not used for the same cases. Temporal electrodes were used with alternating current, while with direct current one electrode was placed on the left temporal region, the other at the top of the heart.

In assembling the results of electroencephalographic investigations by a number of authors it is possible to bring out several facts important from a practical standpoint.

1. There is a definite connection between the pre-shock and post-shock characters of the encephalogram. Patients whose pre-shock electroencephalograms show epileptoid features produce after shock a more modified curve of electrical activity than patients whose pre-shock electroencephalograms are normal.

2. In general the degree of electroencephalographic change varies with the number of induced shocks and is inversely proportional to the interval of days between the most recent

shock and the recording of the electroencephalogram. However, individual differences occur which are due to different reactions by the cortex to the shock.

3. There does not exist any definite correlation between the threshold level of electroshock, that is, the number of shocks required for effecting a change in the electroencephalogram, and the time necessary for establishing these changes after the most recent shock. In other words, the reaction of the patient's brain is conditioned by its resistance.

4. An improvement in the biocurrents of the brain as compared with the condition preceding shock was observed in electroshock therapy for 49 percent of cases as opposed to 30.5 percent for a control group where other methods of therapy were used.

5. Of the patients who had shown improvement, in 75 percent this improvement was either permanent or showed progressive recovery upon reexamination from four months to a year later.

6. It was impossible to make any fundamental differentiations in the electroencephalograms of patients who had shown reactions to the therapy on the basis of clinical findings and those who had not, that is, there was no direct relationship between the dynamics of the electroencephalograph and the clinical picture.

All these findings indicate that success in electroshock therapy depends principally on the stage of the ailment and the depth of the schizophrenic process. Furthermore, the

electroencephalogram helps to determine which patients require such treatment and thereby makes it possible to establish earlier the effectiveness of such therapy.

From the time that convulsion therapy was first put into practice, a number of works have been published stating that treatment by this method in the case of some patients had been observed to be followed by spontaneous epileptic attacks several months following termination of treatment. It is true that such cases have been described not only following electroshock but also other types of shock therapy (metrazol and insulin). All these cases were examined electroencephalographically (but only after therapy) and gave rise to curves quite characteristic for epilepsy. Patsella and Barrera (1945) obtained for two patients with epileptic seizures following electroshock therapy electroencephalograms made prior to therapy and after the occurrence of the seizures. The graph made before therapy disclosed obvious pathological features. Because of this the authors offer the hypothesis that spontaneous epileptic attacks occur only with patients who manifest a predisposition toward convulsions. However, the qualification should be made that the graphs the authors show are not characteristic for epilepsy and frequently are to be met with in cases of true schizophrenia.

At the Third All-Union Congress of Neuropathologists and Psychiatrists a number of reports and talks were devoted to electroshock therapy. However, the problems of the way this method operates and of its effectiveness are still unsolved, especially since these authors did not use controls for therapy

by means of electroencephalographic examination. For this reason we consider it timely to communicate the results of our investigations of graphs of the biocurrents of the brain for electroshock therapy of schizophrenia.

Procedure.

Electroshock was produced by an alternating current apparatus designed by the Central Institute for Psychiatry. Electrodes were attached to both temples (at a distance of 2 centimeters in front of the ear and 2 centimeters above the helix of the ear). The dosage -- 100 to 115 volts. Duration -- 0.5 to 0.8 of a second. Each shock brought on an epileptic-type seizure of general tonic and clonic convulsions including a stoppage of breathing and cyanosis. The tonic convulsions continued for 3-5 seconds, the clonic for 15-20 seconds. For sometime after the shock (up to a half-hour) a deafened condition prevailed. Retrogressive amnesia was noted after every seizure.

Electroencephalographic investigations were made with a stylus oscillograph with a single stylus and a four-cathode booster. Records were taken from the occipital, frontal, and parietal regions on both sides. Recordings were made immediately before electroshock and in some cases immediately after it (for a period of 15-60 minutes), on the day following the fifth or sixth electroshock, and, finally 20-30 days after the final electroshock. Unfortunately, it was outside the control of the laboratory to make recordings of electroencephalograms for longer periods following treatment. Observation is still going on in some cases.

Case 1

Patient K., 23 years old. Entered 1 April 1948. Diagnosis: catatonic form of schizophrenia. She had been ill since December 1947. At night she would jump up as if in fear of something. She said that she was going to be killed soon. Complained of certain changes in her face and would look at herself in the mirror in horror. Shortly afterwards she became taciturn, ceased to converse, and refused to take food. Upon entering the hospital she was completely withdrawn, unapproachable, negative. "Waxy flexibility." Mutism. The first electroencephalogram was made when she was in such a condition. First electroshock took place on 14 June 1948. On 24 April following her third electroshock she lay motionless on her bed. Mutism, negativism. In reply to questions she would whisper something. She had to be fed by hand by attendants. On 8 July she had her fifth electroshock. She remained difficult to approach and self-enclosed. She gave monosyllabic answers to questions, kept to herself, and did not attempt to make contact with other patients. Afflicted with paramimia, sluggishness. On 10 October 1948, after 6 treatments of electrosleep, she was in a state of catatonic stupor, lying inert, not answering any questions, and negativistic. On 23 October, after her eighth electroshock, she became more tractible. She answered questions with a smile, but did not speak. She did not get out of bed. She was fed by hand. On 23 November, after her fifteenth electroshock, she became approachable, affable, emotionally depressed, and expressed broken delirious ideas. She received a total of 17 electroshocks,

the last on 10 December. She was discharged with an apathetic abulic defect in a not very acute form.

Electroencephalography.

On 14 June 1948 prior to electroshock recordings displayed sharp fluctuations in the amplitude of alpha waves varying from 60 to 180 microvolts and having a frequency of 10 oscillations per second. There were frequent periods when the alpha waves disappeared and were replaced by beta waves. Frontal attachments showed alpha waves with low amplitude and a frequency of 12 per second. There were many slow waves with flat crests with a duration of 0.3-0.4 of a second.

Occasional alpha waves were registered by all lead-off wires 35-60 minutes following electroshock.

There were many slow waves with a duration of 0.2-0.3-0.5 of a second with peaked crests and an amplitude of 80-120 microvolts. Typical epileptoid discharges and jointed-like oscillations with an amplitude of 200 to 350 microvolts. Groups of high frequency of 60 oscillations per second with an amplitude of 60-100 microvolts.

On 25 June, after her third electroshock many jointed-like oscillations were to be noticed with an amplitude up to 300 microvolts, especially from the left frontal lead-off wire. There were slow waves with a duration of 0.3-0.4 of a second. Alpha waves were very infrequent.

On 29 July after the fifth electroshock, the alpha rhythm was better expressed than in preceding recordings, although it was very uneven and frequently interrupted by slow waves.

On 22 October after the eighth electroshock, alpha waves were infrequent varying in frequency from 8 to 12 per second. They were frequently interrupted for periods of 0.6 to 0.8 of a second. The alpha waves had an amplitude of 20 microvolts. Frontal lead-off wires disclosed many slow waves.

On 27 December, seventeen days after the final (seventeenth) electroshock, there were many individual and grouped jointed-like oscillations for all wires. They were more sharply expressed when coming off the temporal-frontal wires, especially on the right side. Frontal wires showed very infrequent slow waves lasting for 0.3-0.6 of a second. Other wires did not disclose them. Alpha waves were found to be considerably better than in previous recordings, but they were uneven in rhythm, especially in amplitude, fluctuating from 20 to 40 microvolts.

When first treated by electroshock, the patient showed sharp changes on the electroencephalogram, which spoke of a considerably heightened excitability of the brain. The clinical picture showed no displacement. After the fifth electroshock, when there was a changeover to treatment by electrosleep, electroencephalographic changes and clinical findings increased rather than decreased. However, after a subsequent return to electroshock therapy, a decrease in catatonic stupor began to be noticed, while the electroencephalographic picture began to

resemble a somewhat normal one. After therapy, slow waves almost completely disappeared, while the alpha rhythm became more regular. Manifestations of irritation in the form of jointed-like oscillations still remained.

Case 2

Patient Ye., 23 years old. Was accepted 1 June 1948. Diagnosis: Catatonic form of schizophrenia. Beginning of ailment -- May 1948. Was sick for a long time with malaria; treated with acrichin. Condition prior to therapy: tense, withdrawn, negative; would not accept food.

On 25 October, after having received her 15th electroshock, she was formal, somewhat withdrawn, capricious, and would make faces.

On 16 November, after having received 25 electroshocks, she was much more responsive and spent all her time either reading or making translations from German. She was selectively social with doctors, but discourteous and demanding with respect to attendants.

All recordings on the electroencephalogram for the various wires showed an uneven alpha rhythm, each wave having a duration of 0.06 to 0.15 of a second and an amplitude of 10 to 50 microvolts. In places the amplitude of the alpha waves would decrease to the extent of total disappearance, where it would be replaced by beta waves. These changes were particularly marked for the frontal regions where alpha waves of low amplitude and many slow waves lasting 0.4 to 0.6 of a second prevailed. There was a

marked change in the picture after the fourth electroshock. Alpha waves could not be seen for the occipital areas. The graph disclosed as a primary feature slow waves lasting 0.4-0.6-0.8 of a second on which beta waves were superimposed. Quite frequently individual and grouped jointed-like oscillations were to be found reaching an amplitude of 100-120 microvolts. Upon termination of therapy these jointed-like oscillations had disappeared, but there remained a small number of slow waves of very uneven amplitude and duration (Figure 1).

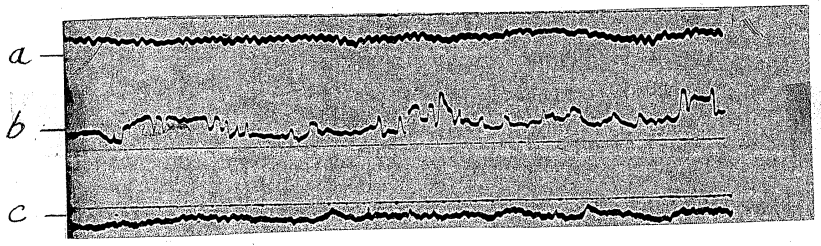

Figure 1. Patient Ye. (a) prior to electroshock, (b) after the fifth electroshock, and (c) three weeks after the final (25th) electroshock.

Case 3

Patient S., 27 years old. Diagnosis: catatonic paranoid type of schizophrenia. He had become ill in March 1946, turning silly, awkward in behavior, laughing without cause. At times he refused food and would not talk. In November and December he manifested motor excitement. At that time he was in the First Suburban Psychiatric Hospital. On 27 May 1948 he made an attempt at suicide by wounding himself in the vicinity of the heart, after which he talked deliriously of having to be put in prison, of

being watched, etc. From June 1948 he raved about being poisoned, refused food, and stopped conversing. First electroshock was administered 2 November. On the next day he displayed complete mutism, lay in bed, ate well, went by himself to the latrine. After having received ten electroshocks, his electroshock therapy was terminated on 22 November. From 14 December he began to talk again, and again started to rave about being poisoned, that is, he was being poisoned through milk; this was the reason that had made him originally ill. Courteous. Somewhat silly. Inclined toward humor. Would write many letters of a fantastic nature.

Electroencephalography

On 2 November 1948 attention was drawn to the very low amplitude and occurrence of alpha waves even for the occipital regions. Their amplitude hardly reached 15 microvolts. The beta rhythm was particularly marked and present in all potentials. The electroencephalogram showed a marked change after the sixth electroshock. Almost the entire potential (for the left occipital region) consisted of large slow waves lasting 0.6-0.8 of a second on which there were superimposed single alpha waves reaching the high frequency of 70 a second. In places there were to be noticed jointed-like oscillations for the potential having an amplitude up to 100 microvolts. Three weeks after termination of electroshock therapy, the slow oscillations on the electroencephalogram disappeared, while the alpha waves were very irregular in amplitude and frequency. There were considerably fewer beta waves than in preceding recordings.

Thus electroshock therapy was effective only in removing mutism and in making the patient more responsive. The electroencephalogram again became pathological, similar to the way it had been prior to therapy (Figure 2).

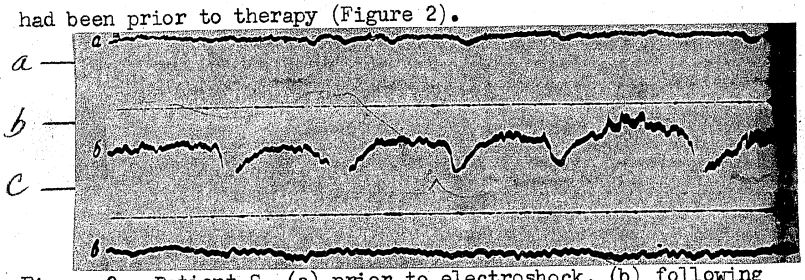

Figure 2. Patient S. (a) prior to electroshock, (b) following sixth electroshock, (c) 20 days after final electroshock.

Case 4

Patient M., 17 years old. Was accepted 10 February 1948. Diagnosis: schizophrenia. He had become ill during the autumn of 1947. Showed lack of self-confidence and manifested suspiciousness. Quit school. His condition deteriorated from January 1948. Had auditory hallucinations, complained of depersonalization. Electroshock therapy was begun on 11 May. His condition remained unchanged after the first electroshock. On 15 May the second electroshock was administered. After this he became apathetic, irritable, and showed lack of self-control. Depersonalization not so marked, would not speak spontaneously of his fantasies. The patient was transferred to insulin therapy.

The registration on the electroencephalogram prior to electroshock was marked for all regions by irregular frequency and amplitude of the alpha rhythm. Moreover, low amplitudes, not

exceeding 30 microvolts, prevailed. In places alpha waves would disappear and be replaced by beta waves for periods of 1-2 seconds. The latter were quite clear and superimposed on alpha waves. Slow waves were to be found very rarely. Six hours after the first electroshock, which was accompanied by a convulsion seizure, the lines on the electroencephalogram sharply changed. Slow waves appeared with acute vertices; sometimes they assumed the character of acute epileptoid waves. Alpha waves were not to be seen. Twenty-four hours after the second electroshock the potentials of the encephalogram assumed the same character they had prior to treatment. Only an increase in the number of slow waves with a duration of 0.6-1.0 second could be perceived.

Sharp changes were to be noticed on the electroencephalogram immediately following each electroshock, evidence of the sharp increase of excitability in the brain; however, the electroencephalogram lines finally reassumed their original appearance.

Case 5

Patient P., 26 years old. Was admitted 28 August 1948. Diagnosis: catatonic form of schizophrenia. She had become ill during the summer of 1947. Condition before entrance: withdrawn, self-enclosed; would make stereotyped movements with her hands; impulsive. Would eat only at the insistence of attending personnel. She had had auditory hallucinations upon first becoming ill. Became much more active after insulin therapy. She would get up out of bed, eat in the dining room, read, listen to the radio, be quite responsive. First electroshock administered on 6 October. After

the sixth electroshock, she became capricious, careless, untidy, unapproachable, and wet her bed. Speech became disconnected. Impulsive. Frequent motor excitement. Erotic. Had visual hallucinations.

Electroencephalography

On 6 October 1948 there was a very irregular and unstable alpha rhythm. There were many high beta waves replacing alpha waves. High frequency (65-70 per second). Their amplitude varied sharply from 33 to 100 microvolts. Changes were most marked in the temporal and frontal regions.

Following her sixth electroshock on 18 November many slow waves in all regions were noticed, the majority having acute vertices, and groups of jointed-like oscillations with an amplitude of 60-70 microvolts. Alpha waves occurred in isolated instances even in occipital areas (Figure 3).

In this instance the fact is to be noted that the electroencephalogram prior to electroshock, that is, after termination of insulin therapy, was most variable and showed signs of cortical excitability despite clinical improvement and relatively quiet behavior. After the sixth electroshock, signs of excitation on the encephalogram were sharply intensified; this was accompanied by clinical deterioration. The electroencephalogram was thus instrumental in warning of care in the use of electroshock, which is why it was discontinued after the sixth time.

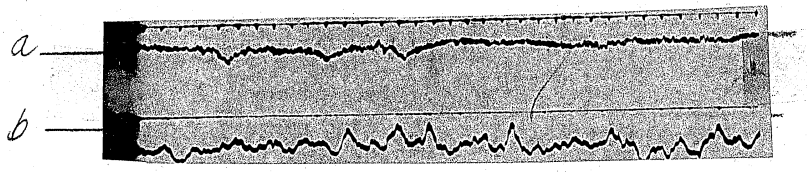

Figure 3. Patient P. (a) potential on electroencephalogram prior to electroshock therapy, (b) following sixth electroshock.

Case 6

Patient D., 21 years old. Was admitted on 30 November 1948. Paranoid form of schizophrenia. She had been ill since she had been seventeen years of age. Studied poorly; was dull and apathetic. Evidenced auditory hallucinations, negativism, clumsy behavior. Improved after six weeks, began to study. In 1946 she had a relapse with the same characteristics. This time she came out of it incompletely, as she could no longer study. In 1947 she had a second relapse of the same character. She was treated with insulin, after which her condition improved. There was a third relapse in September 1948. Onset of auditory hallucinations, worry, aggressiveness. She received insulin therapy from 9 October to 20 October. On 1 November she was dull, withdrawn; at times would display delirious attempts at resistance. First electroshock administered on 6 December, second on 9 December, and the third on 13 December. Following the electroshocks there was no noticeable change in the psychic condition of the patient. She was dull, apathetic, and abulic.

Electroencephalography

1 November 1948, prior to electroshock: irregular alpha

rhythm; low amplitude varying between 15 and 30 microvolts. There was a small number of slow waves lasting 0.4-0.6-0.8 of a second on which alpha waves were superimposed. A large number of small beta waves. These modifications were more sharply marked in the frontal areas.

On 14 December, after three electroshocks, a considerable increase took place in the number of slow waves lasting 0.4-0.6-0.8 of a second, a large part of which had sharp vertices; alpha waves of irregular amplitude were superimposed on them. Beta waves were weak.

Alpha waves of low amplitude and an abundance of slow waves are to be noticed. No jointed-like [spike-like ??] oscillations of the potential were observed as in other cases of electroshock therapy.

Case 7

Patient Zh., 44 years old. Was admitted 28 August 1948. Diagnosis: paranoid hallucinatory form of schizophrenia. She became ill in 1940. Was treated with insulin, after which she went back to work. In 1941 another attack occurred accompanied by delusions of persecution, auditory hallucinations. She was given insulin therapy a second time with successful results. Returned to work. A third attack took place in 1948: delusions of persecution, hypnotic reactions, auditory hallucinations in which the radio suggested strange thoughts to her. Undertook a course of insulin therapy. On 25 November, immediately prior to the beginning of electroshock therapy, she was dull, considerably withdrawn, and apathetic. At times she would give utterance to

broken delirious ideas of resistance and suggestions of thoughts, and have hallucinations. Six electroshocks in combination with insulin shock were administered between 26 November and 16 December. All electroshocks caused only a short loss of consciousness with slight distortions of the face.

Electroencephalography

On 25 November 1948 there was an unsteady and irregular alpha rhythm, most sharply noticeable in the temporal and frontal areas on both sides. Slow waves lasting 0.6 to 1.8 seconds were found in the frontal areas. Beta waves were most sharply expressed in intervals between alpha waves.

On 24 December after 8 electroshocks alternating with insulin shocks, the alpha rhythm was seen to be very uneven and unsteady. It would disappear for 2-3 seconds and be replaced by high beta waves. Many jointed-like oscillations and steep waves were to be noticed, especially in the frontal areas.

On 27 December a second recording of the electroencephalogram could not be made because of marked negativism on the part of the patient. The procedure of recording the electroencephalogram was met with delirious resistance.

Only tentative conclusions may be drawn because of the small number of patients that have been examined.

There is no doubt that electroshock sharply increases the excitability of the brain. This is evidenced by the appearance of sharp waves, jointed-like oscillations, and even epileptoid

terminate treatments. In electroshock therapy modifications of the electroencephalogram can serve as an adequate control agent.

Despite the small number of patients so studied, it can now be said that reactions of electroencephalograms are not identical for different patients in electroshock therapy. In conformance with clinical findings this reaction and the dynamics of the electroencephalogram vary from case to case. Evidently much significance with respect to the character of this reaction is to be attached both to the overmorbid condition of the brain and the general somatic condition of the patient.

Durability of electroencephalogram changes noticed many days after administering of electroshock speaks of the considerable injury to the brain occurring because of this type of therapy. Such a consequence of electroshock speaks for the necessity of applying it most carefully and in doses which do not bring on aggravated convulsion seizures.

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